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ATYPICAL DEVELOPMENT

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PSYCHOLOGY TEACHERS UPDATE

Psychology Teachers Update is designed to give a brief overview of the main developments in the different areas of psychology. There is a proliferation of journals and research, and it is very difficult to keep abreast of the latest trends, particularly in the many and varied areas of psychology.

Each issue of Psychology Teachers Update will cover a particular topic, and summarise the main research directions and findings in the last ten to fifteen years approximately. The aim is to give teachers the feel of what is happening in that area of psychology.

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PAST ISSUES

No.1 - September 2002: Memory
No.2 - January 2003: Evolutionary Psychology
No.3 - May 2003: Biological Psychiatry
No.4 - September 2003: Social Constructionism

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INTRODUCTION

> Dyslexia is no longer a specialist medical term - it has been thoroughly adopted into everyday language. Everyone knows that dyslexics are not able to read properly or spell in a weird fashion. Dyslexics themselves talk poignantly about their difficulties with written language.. (Frith 1997 p1).

This article focuses upon developmental dyslexia which appears in childhood as opposed to acquired dyslexia as a result of brain injury or disease.

"Dyslexia" (Latin for "difficulty with words") was first coined by a German doctor, Dr.Berlin in 1887. James Hinshelwood (a Scottish eye surgeon) reported cases of "word blindness" in 1895. Samuel.T.Orton (an American neurologist) proposed the first theory in 1925.

DSM-IV estimates that developmental dyslexia affects 4% of the population (APA 1994), though figures from other studies vary between 5-17% (Shaywitz 1998).

Reading and learning difficulties receive more attention today for a number of reasons: most importantly, because schooling is compulsory in many countries, but also that the highly competitive employment market makes educational qualifications paramount (Selikowitz 1998).

DSM-IV, the classification system of mental disorders from the American Psychiatric Association (1994) does not name dyslexia, but talks about "Reading Disorder" (315.00). The criteria for diagnosis are:

> A. Reading achievement, as measured by individually administered standardised tests of reading accuracy or comprehension, is substantially below that expected given the person's chronological age, measured intelligence, and age-appropriate education.
>
> B. The disturbance in A significantly interferes with academic achievement or activities of daily living that require reading skills.
>
> C. If sensory deficit, reading difficulties in excess of usually associated (APA 1994 p50).

While the WHO (1992) classification of mental disorders, ICD-10, has two categories relevant here: "Dyslexia and Alexia" (R48.0) and "Specific Reading Disorder" (F81.0).

It is important to note that a "reading disorder" is different to "reading backwardness". The latter can be seen in general developmental delays in the individual, overt neurological disorders, or social disadvantage. It is simply a slower progress in reading. A "reading disorder" is shown in qualitatively different ways.

Characteristics of Dyslexia

Individuals with dyslexia show a number of characteristics which are more than just problems in learning to read (table 1).

- Problems in understanding letter-sound correspondences
- Poor spelling
- Problems with written language
- Poor short-term memory
- Poor mental arithmetic
- Difficulties in naming familiar objects and learning labels for new objects
- Problems with learning sequences (like months of the year)
- Problems with verbal information

(After Wood and Richardson 2002)

Table 1 - Characteristics of dyslexia other than problems with reading.

Though the emphasis in diagnosis is upon reading problems, difficulties in spelling are also important indicators of dyslexia. Table 2 shows the spelling difficulties of a boy called "J.M" (Snowling 2000).

TARGET WORD	AGE: 8yrs	10yrs	12yrs
umbrella	unenprl	unbrl	unberller
understand	unenstand	understant	unstand
cigarette	sikeoleg	cigeragg	citterlit

Table 2 - Examples of spelling attempts by "J.M" at different ages.

Because of the variety of problems experienced by dyslexics, it may be that there are sub-types which give rise to "dyslexias". Boder (1973), based on reading tests, distinguished dysphonetic dyslexia (the main problem with phonetic analysis of words), and dyseidetic dyslexia (the main problem here with the visual shape of words). Snowling (2000) explores the research on phonological (problems sounding out unfamiliar words), surface (unable to recognise words, except by individual letters, and confused sounds eg: "saw" and "sore"), and

deep dyslexia (eg: read "lion" as "tiger").

Most of the emphasis is upon the problems related to dyslexia, but there are advantages which are often overlooked in a literacy-based society. For example, good visual and spatial skills (as used in engineering).

CAUSES OF DYSLEXIA

Historically, and currently the theories of dyslexia proliferate. From brain damage or malformation, to maturational lag, cerebral dominance, or information processing deficits among others (Selikowitz 1998).

Recent theories to explain the causes of dyslexia, including low-level deficits in the visual system (eg: Lovegrove 1991), impairments in the formation of general cognitive skills (eg: Nicolson and Fawcett 1990), and deficits in the control of eye movements (eg: Pavlidis 1983).

However, many researchers feel that more promise exists in seeing dyslexia as a linguistic or phonological deficit. But even this idea has sub-divisions of theories. It may be due to meta-linguistic problems (decomposing spoken words into phonological elements) (eg: Byrne and Fielding-Barnsley 1990); or problems in speech perception (eg: Brandt and Rosen 1980); or due to general language dysfunctions (eg: Aram and Norton 1980). Locke et al (1997) explored the different theories in an extensive longitudinal study of children born to dyslexic parent(s).

Frith (1997) offers a model of the causation of dyslexia that combines a number of factors. This model includes causes at the biological and environmental level; the behavioural level which gives the observed behaviour; and the cognitive level which links the biological and behavioural levels (figure 1).

BIOLOGICAL LEVEL

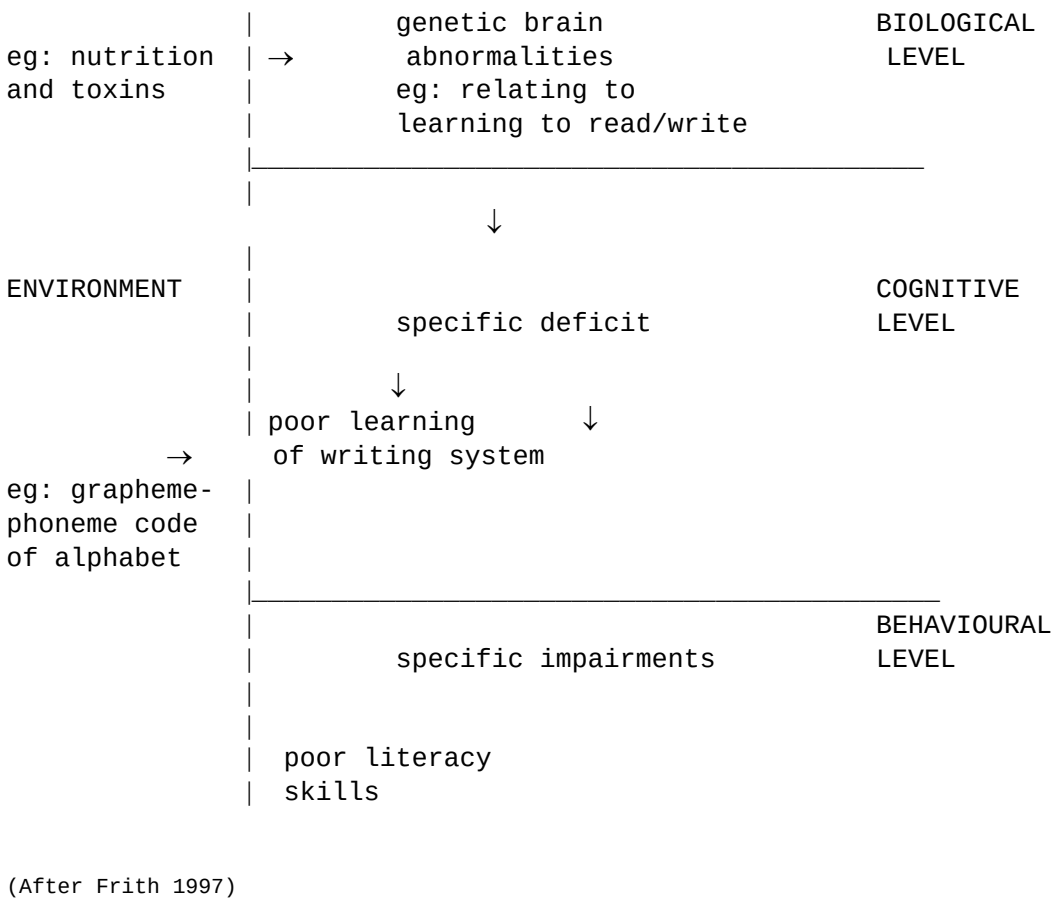
Hemispheric Brain Differences

The search for an underlying physiological cause to dyslexia includes abnormal symmetry in the structure of the planum temporale in the brain (Galaburda 1989)

The brain abnormalities being found with dyslexia are seen as due to variations in brain development in the womb (Galaburda 1993). Problems with the sulci and gyri

(folds and grooves on the surface of the brain) [1] which are signs of intra-cortical connections linked to cognitive and behavioural deficits (Hynd and Hiemenz 1997). These differences can be seen in hemispheric symmetry.

In the general population, language abilities are specialised in the left hemisphere (hemispheric asymmetry). Dyslexia could be due to a lack of asymmetry or specialisation, or to the reversed asymmetry (ie: language in the right hemisphere) (Hier et al 1978).



(After Frith 1997)

Figure 1 - A causal model to explain dyslexia.

Before the development of imaging techniques, the post-mortem study was the main research tool for assessing brain differences. For example, Galaburda and Kemper (1979) found symmetrical plana temporale in the brain of a man with developmental dyslexia who died from an accident. In the general population, the left planum temporale is usually larger.

[1] Research has looked at types 1-4 in terms of sulcal and gyral patterns in the perisylvian region (details in Hynd and Hiemenz 1997).

Galaburda et al (1985) confirmed earlier findings in three other post-mortem brains, as well as cell anomalies in the perisylvian area of the left hemisphere. Galburda (1994), in other post-mortems, has found more microscopic anomalies in the right hemisphere of dyslexics. These anomalies include ectopias (clusters of neurons in unusual locations of the cortex), and micropolygyrias (excessive cortical folding).

These differences would have occurred during the middle of gestation at the time of cell migration (when cells are allocated to the correct area of the growing brain of the foetus). This leads to the production of unusual patterns of connectivity in language-related regions of the temporal cortex (Rosenzweig et al 1999). But Schultz et al (1994) found no such differences in the brain when controlling for brain size, age, and gender.

With the development of Magnetic Resonance Imaging (MRI) technology [2], the findings of brain differences in dyslexics have been confirmed with living patients; eg: Larsen et al (1990) found plana symmetry in 70% of 19 dyslexics compared to 30% of 18 control children.

Imaging technology has also found other brain differences; eg: Shaywitz et al (1998) used Functional MRI (fMRI) during a reading task. Dyslexics had little activation of the posterior regions (including Wernicke's area), and an overactivation of the anterior regions of the brain. Demb et al (1998), in a similar experiment, found less activation of the visual cortex to written words in dyslexics.

Genetics

The discovery of a genetic basis to reading problems would give more support to the biological basis of such behaviour. There are a growing number of twin studies in this area. Table 3 summarises three of the main studies.

More sophisticated statistical analysis has made use of multiple regression analysis to produce a "discriminant function score" (composite measure of reading and spelling performance). Analyses like these have found that about half of the difference between twins in reading ability is due to "heritable influences", but that "genetic factors may be less important as a cause of reading/spelling deficits in older children.." (DeFries et al 1997 p31).

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[2] Details of neuroimaging techniques in "Biological Psychiatry", Psychology Teachers Update no.3, May 2003.

The search for the exact genetic origin of dyslexia has focused on genes on chromosomes 15 (Smith et al 1983), 1 (Rabin et al 1993), and 6 (Cardon et al 1994).

STUDY	NUMBER OF PAIRS	CONCORDANCE RATE	NOTES
Bakwin (1973)	31 MZ 31 DZ	91% 45%	Sample from interviews with 338 same-sex twin pairs; definition used: "reading level below the expected derived from child's performance in other school subjects"
Stevenson et al (1987)	14-19 MZ 27-43 DZ	38-50% 54-43%	Sample from 285 pairs of 13 year olds in London; use of standardised tests eg: Schonell Graded Word Reading and Spelling Tests (Schonell and Schonell 1960); "reading backwardness" = reading age greater than 18 months below chronological age; "spelling backwardness" also studied
DeFries et al (1997)	195 MZ 145 DZ (same sex)	0.67 0.37	Colorado Twin Study of Reading Disability

Table 3 - Results of three twin studies of dyslexia.

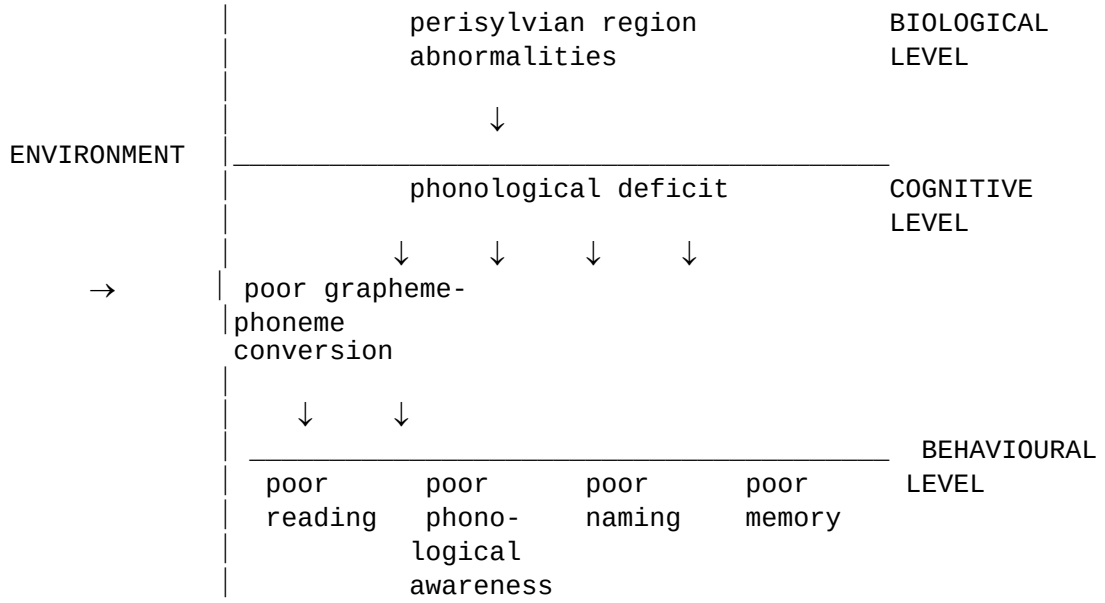
COGNITIVE LEVEL

Ramus (2003) sees the "dyslexia scene" as occupied by four major theories which are grouped into two "antagonistic frameworks". One side of the debate focuses upon a cognitive deficit related to the processing of sounds (known usually as the phonological deficit hypothesis). The other side of the debate sees a general sensori-motor deficit as most important. This deficit may be caused by an auditory deficit, a magnocellular visual dysfunction, or a cerebellar/motor dysfunction.

Dyslexia as Single Problem - Phonological Deficit Hypothesis

The consensus among researchers is that the central difficulty in dyslexia is related to processing of speech sounds, though other deficits may also exist (Temple 2002).

This theory focuses upon problems with speech sounds as the basis to dyslexia. A specific cognitive deficit like this one affects reading through the interaction with an alphabetic writing system (Morton and Frith 1995) (figure 2).



(After Frith 1997)

Figure 2 - Model of phonological deficit hypothesis and dyslexia.

Problems with phonological awareness can be linked to learning to read in that the process of reading involves understanding how speech sounds are represented by letters. In other words, how to translate between written and spoken language. The "natural" process of speech segmentation (dividing language into phonemes) in language acquisition does not automatically lead to the mapping of sounds to graphemes in reading.

But Goswami (1997), because of lack of research, is unsure about whether dyslexic children are following the same sequence of phonological development as non-dyslexic children at a slower rate.

The underlying biological basis to the phonological deficit is seen as an abnormality in the perisylvian region of the left hemisphere (which divides parietal and frontal lobes from temporal lobe, and includes Broca's and Wernicke's areas) (Galaburda 1989).

Paulesu et al (1996), using PET scans, compared five adult dyslexics, and university graduates of the same age as controls. The tasks were rhyme judgement (eg: does "T" rhyme with "Z") which required internal evocation of the speech sounds, and visual memory (eg: recall of target letter). The dyslexic participants showed less blood flow activity in the left hemisphere around the perisylvian fissure. This is called the left temporoparietal cortex area in some studies.

Subsequent neuroimaging studies have focused the area of dysfunction (eg: area BA 37; Temple 2002). But

most of the studies are with adult dyslexics, and the observed brain patterns may reflect a "lifetime of compensation" (Temple 2002). Temple et al (2001) is one of the few neuroimaging studies with dyslexic children. A fMRI study of 24 dyslexic and 15 normal reading children (8-12 years old) confirmed the findings from the adult studies.

The existence of a phonological deficit can be shown from behaviours by dyslexics:

- Slow at rapid automatic naming (RAN): naming familiar objects under time pressure;
- Poor verbal short-term memory, but normal memory span for visual information;
- Poor at word and non-word repetition; eg: "hopital" for "hospital";
- Poor phoneme awareness; eg: "buttercup" = "but", "er", and "cup";
- Difficulties in object naming: the explicit retrieval of verbal information from long-term memory;
- Impairment in rhyming, syllable counting, sounding pseudo-words

Longitudinal studies (eg: Scarborough 1990) have shown a common basis between language acquisition and reading problems in that children later diagnosed with dyslexia showed language problems at two years old.

There is some debate over whether the phonological deficit has an underlying cause related to the processing of rapidly changing auditory information (rapid processing hypothesis; Tallal 1980). Deficits in the processing of sound rapidly impairs the ability to discriminate auditory cues related to distinguishing phonemes. This dysfunction is probably located in the left prefrontal cortex. The debate is framed in Ramus (2001) and Temple et al (2000).

Interestingly, Temple et al (2000) claimed to have reduced the effects of this disruption by training.

Dyslexia as Single Problem - Auditory Deficits

These theories relate to problems in the brain with the processing of sounds. Auditory processing deficits in dyslexics has been confirmed by different methods; eg: discrimination of frequency and intensity of tones.

For Ramus (2003), there are three questions that need answering:

i) What proportion of dyslexics are affected by an

auditory processing deficit? Not all would be the answer: 45% in one study (Tallal 1980), and from all studies, 39% (Ramus 2003).

ii) Is the auditory processing deficit the same as a deficit in rapid auditory processing? Rapid auditory processing relates to short sounds and fast changes in sound. Studies suggest that they are not the same thing.

iii) Does the deficit in auditory processing explain the phonological deficit? Generally this does not seem to be the case. An auditory processing deficit exists only in a sub-set of dyslexics.

Paulesu et al (1996) noted unusual brain activity patterns in their neuroimaging study. Dyslexics, during phonological processing, showed no activity in the posterior language brain areas, but activity in the left frontal language areas. During rapid auditory processing, there is also no activity in the left prefrontal brain areas. These patterns of brain activity could be called a "disconnection syndrome".

Such a pattern of activity would be the product of disruptions of white matter function in the brain (ie: cell connections). Klingberg et al (2000) were the first to use diffusion tensor imaging (DTI) to confirm this idea. DTI is a variation of MRI which looks at the microstructure of the brain.

Dyslexia as Single Problem - Visual Deficits

The emphasis for this group of explanations is upon problems in the processing of visual information in the brain.

Ramus (2003) again frames the debate here in three questions:

i) Do visual disorders cause reading difficulties? Minor visual problems are "plausible proximal causes of reading impairment" only (Ramus 2003).

ii) Do the visual disorders have a magnocellular cause? The evidence here is unclear.

iii) What proportion of dyslexics have such a visual deficit? It is only a sub-set of dyslexics who have visual problems.

The visual pathways through the brain from the retina to the visual cortex include layers of large cells known as magnocellular cells (as well as small

parvocellular cells). The magnocellular system appears to respond to particular types of visual information (eg: lower spatial frequency and higher temporal frequency stimulation), and to the onset and offset of stimuli (Hogben 1997). Dyslexics seem to have deficits in the magnocellular system according to supporters of the visual deficit idea.

Research by Martin and Lovegrove (1988), for example, found that dyslexics had lower contrast sensitivity. This is a poorer ability to distinguish the contrast between two stimuli. But there is not full agreement about the evidence (Hogben 1996).

A problem exists in trying to explain the link between the magnocellular deficit and reading problems. One possibility is known as "transient-on-sustained inhibition" (Breitmeyer 1980). Simply, as the eyes move across the printed page, the contents of the last eye fixation are carried into the next fixation, thus causing confusion in what is seen. The sustained system in attention is not inhibited after each fixation.

While the "temporal precedence theory" (eg: Williams et al 1987) sees the analysis by the magnocellular system of visual information as too slow at passing information to the parvocellular system. This means that the progression of visual information to the processing stage is interfered with. The evidence for both these theories is discussed in Hogben (1997).

Dyslexia as Multiple Problems

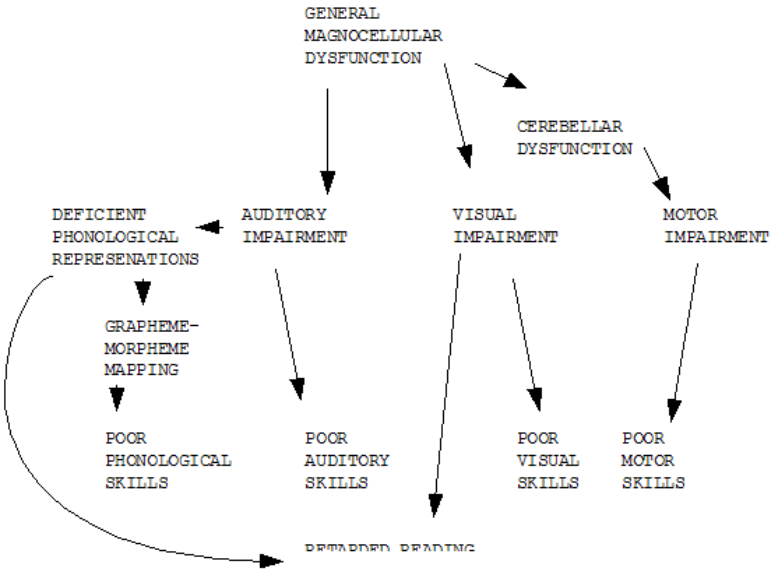
Because dyslexia is more than just a problem with reading, some theories have suggested that the cause is due to more than one factor. For example, Eden et al (1996) see the cause of dyslexia as a phonological deficit and a visual deficit. This latter deficit can be seen in poor motion-detection. The origin of each of the deficits would be different brain abnormalities.

While Nicolson and Fawcett (1995) suggested that a single brain abnormality, in the cerebellar area, could produce a phonological deficit and a motor control deficit. The motor control deficit is seen in poor time estimation and poor balance, and also interacts with the phonological deficit in poor naming speed. The evidence for this theory is summarised in Nicolson et al (2001). Ramus (2003) suggests that the evidence is limited though.

Other combinations include problems in auditory, visual, and tactile processing (Laasonen et al 2002).

Stein (2001) has attempted to combine the different

theories with the general magnocellular theory of dyslexia, which views the cause as phonological and visual deficits (figure 3).



(After Ramus 2003)

Figure 3 - The general magnocellular theory of dyslexia.

Conclusions on Cause of Dyslexia

There is a lot of research, particular neuroimaging, in recent years into the cause of dyslexia. But none of the research is conclusive about the causality of dyslexia:

> It is not clear whether, for example, the disorder in white matter organisation comes first and leads to the disruptions in behaviour and brain function related to rapid auditory and phonological processing. Alternatively, do deficits in the brain related to rapid auditory processing comes first and then lead to disruptions in phonological processing which, in turn, affect the organisation of temporoparietal white matter? (Temple 2002 p181).

However, there is generally little disagreement that dyslexics have brain differences in the left hemisphere mainly.

The many different theories of dyslexia probably

describe the sub-sets of dyslexia, whereas a phonological deficit is evident in all dyslexics studied (Ramus 2003). But there is still a need for a general theory which can "explain both the neurological origin of the specific phonological deficit, and the reasons why a sensorimotor syndrome occurs more often in the dyslexic than in the general population" (Ramus 2003 p216).

TRAINING PROGRAMMES

Phonemic Awareness

Training programmes that focus on the development of phonemic awareness do show improvements in young children.

Borstrom and Elbro (1997), for example, used 136 children from Danish kindergarten classes for their study (average age 6 years and 3 months old). There were three conditions in the study: (i) 36 children of dyslexic parent(s) who received a programme to help develop phoneme awareness ("at risk experimental group"); (ii) 52 children of dyslexic parent(s) who served as a control ("at risk control group"); and (iii) another control group of 48 children of "normally reading parents".

The training programme focused on teaching each sound to the child in a variety of ways, and lasted 30 minutes each school day for 17 weeks. Tables 4 and 5 show a summary of the main findings.

	AT RISK EXPT	AT RISK CONTROL	CONTROL
Phoneme awareness composite score* (out of 17)			
BEFORE TRAINING	4	4	6
AFTER	8	6.5	10
Syllable awareness composite score** (out of 14)			
BEFORE TRAINING	7.5	8.5	10
AFTER	10	10	12

* For example, "phoneme deletion" task - to say word without a particular sound (eg "mice" and "ice").
** For example, "syllable identification" task - to identify picture that contains particular syllable.

(After Borstrom and Elbro 1997)

Table 4 - Approximate average scores from tests before and after training programme in phonemic awareness.

AT RISK EXPT	AT RISK CONTROL	CONTROL
6 of 36 (16.6%)	21 of 52 (40.4%)	4 of 48 (8.3%)

(After Borstrom and Elbro 1997)

Table 5 - Number of children identified as "possibly dyslexic" based on reading status one year after training programme for phonemic awareness.

Other

Lindamood et al (1997) show a number of techniques to stimulate phonemic awareness through articulatory feedback (see also Snowling 2000). Wise et al (1997) review the success of using "talking computers" to teach phonological awareness.

Other ideas that attract media attention, usually as the "magic solution", include the use of coloured glasses (Bouldoukian et al 2002). While the "New Scientist" (Conference report 2003) recently reported a computer game (invented by Terry Lawton) that strenghtens left-right movement discrimination and consequently improved the reading skills of dyslexic children. This would give support to dyslexia as caused by a deficit in the brain's motion processing pathway (another theory of dyslexia).

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Autism

This article will consider what autism is, it's history, features, prevalence, prognosis, causes and current research.

ORIGINS OF THE TERM AND THE DISORDER

The term autism is derived from the Greek word "autos" meaning "self". It was first introduced by Bleuler in 1911, who used it to describe the social withdrawal seen in adults with schizophrenia.

Frith (1989) speculated that we can find evidence of autism throughout history; for example, the "Blessed fools" of old Russia, who were insensitive to pain, had bizarre behaviour, innocence, and lack of social awareness, may have had autism.

Kanner, an American psychiatrist, made observations in 1938 of a five year old and claimed that there were several features characteristics of all children he saw. In 1943, he separated autism from schizophrenia and produced diagnostic criteria for "autistic disturbances of affective contact", comprising of eleven presenting symptoms that have become known as "Kanner's autism" or "classic autism":

i) disorder present at birth;
ii) inability to develop relationships with others;
iii) developmental delay in speech;
iv) non-communicative use of speech after it's development;
v) delayed echolalia;
vi) pronoun reversal;
vii) repetitive and stereotyped play activities;
viii) insistence of sameness;
ix) lack of imagination;
x) good rote memory;
xi) pleasant physical appearance.

Lorna Wing (1988) suggested that children with "Kanner's autism" are rare and that a continuum/spectrum, including abnormalities of the skills below is more realistic:

a) social interaction;
b) social communication;
c) social imagination;
d) repetitive activities;
e) language;
f) sensory responses;
g) movements;

h) special skills (often known as an "islet of ability"; eg: exceptional musical ability in an individual with a general IQ of 60).

The central core abnormalities, known as the "Triad of Impairments" are (a), (b), and (c) above. The triad can occur in a wide range of manifestations and can be associated with many other impairments, including epilepsy and learning disabilities. It is the presence of these impairments that diagnose autism, in varying degrees according to the individual - hence continuum or spectrum disorder.

There are three fundamental impairments which capture Wing's triad (Happe 1994):

i) qualitative impairment in reciprocal social interaction;
ii) qualitative impairment in verbal/non-verbal communication and in imaginative activity;
iii) markedly restricted repertoire of activities and interests.

A word about Aspergers syndrome: this is a form of autism associated with high intellect and/or ability.

Models to explain autism have been developed and one of the most widely accepted describes the disorder on three levels: biological, cognitive, and behavioural.

Morton and Frith (1995) introduced a specific diagrammatic tool for thinking about levels of explanation in developmental disorders; eg: autism (figure 4).

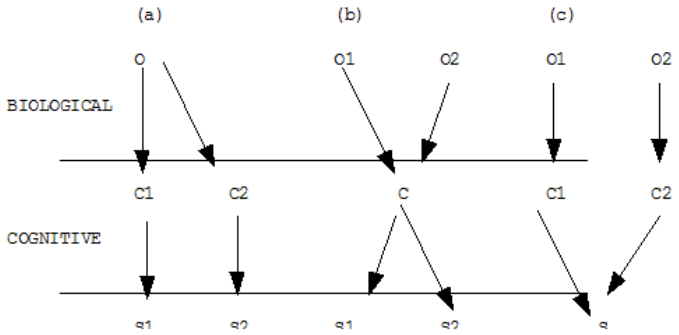

Figure 4 - Morton and Frith's (1995) causal model of three types of disorder.

In figure 4, pattern (a) is the case of a disorder defined by its unitary biological origin (O) which may

have diverse effects at cognitive and behavioural levels. For example, damage or an abnormality in one area of the brain.

Pattern (b) is a disorder with multiple biological causes and several different behavioural manifestations but a single cognitive deficit (C); eg: autism or dyslexia according to some cognitive theorists. While pattern (c) is the case of a disorder defined by behavioural features (S) alone with multiple biological causes and cognitive natures.

CLINICAL DIAGNOSIS OF AUTISM

Autism can be diagnosed in a number of ways including the Diagnostic and Statistical Manual (DSM-IV) (APA 1994) and the International Classification of Diseases (ICD-10) (WHO 1992). Below is the listing from DSM-IV. This is similar to those listed in ICD-10 and focuses upon the Triad of Impairments.

A total of six or more items from (1), (2) and (3) with at least two from (1) and one from each of (2) or (3) (table 6).

1. Qualitative impairment in social interaction

a. Impairment in multiple non-verbal behaviours
b. Failure to develop peer relationships as age-appropriate
c. Lack of spontaneous seeking to share activities/objects
d. Lack of spontaneous social/emotional reciprocity

2. Qualitative impairment in communication

a. Delay or lack of spoken language (and no compensation by other communication)
b. With language, impaired in conversation
c. Stereotypical/repetitive use of language
d. Lack of make-believe play

3. Restricted repetitive/stereotyped behaviour

a. preoccupation with particular activity
b. Inflexible adherence to non-functional routines
c. Repetitive motor mannerisms
d. Persistent preoccupation with parts of objects

Table 6 - DSM-IV criteria for autistic disorder.

Diagnostic and Suggested Prevalence

The age of onset is considered to be 0 to 2 years and 6 months. A reliable diagnosis of autism is rare before age 3 or 4 years as types of behaviours which are impaired in autism don't arise in normal children of this

age (Happe 1994).

However, a study by Baron-Cohen et al (1992) devised a screening checklist for autism in toddlers (CHAT - Checklist for Autism in Toddlers), which focused on pretend play, joint attention, social interest, and social play.

CHAT was used by doctors and health visitors to screen forty-one 18-month-olds, all with an autistic sibling. The results of the study suggested that autism may be detectable at 18 months by looking for deficits in social, communication, and imaginative competence.

The suggested prevalence is half a million in the UK, or 10 per 10 000 children (MRC 2001). There seems to have been a huge surge recently, possibly due to a number of factors: a genuine increase, greater public awareness and/or an increased sophistication in diagnostic measures.

Epidemiology

The numbers of those diagnosed is very dependent upon diagnostic criteria and definition and studies offer a variety of statistics including the gender split. Wing and Gould (1979) suggested 21 per 10 000 for triad measure, and 4-10 autistic children in every 10 000 for "Kanner's autism" (Happe 1994).

There are extreme gender differences that again differ given different diagnostic criteria. Studies suggest that autism affects between five times and twice as many boys than girls (Lord and Bailey 2002).

Females presenting symptoms tend to be at the very high functioning level (Aspergers) or the very low (extreme disablement of the triad and with learning disabilities).

Prognosis

Prognosis refers to the course of the illness. Despite changing definitions, autism remains a severe, chronic developmental disorder that results in significant lifelong disability with a poor prognosis where most people on the autistic spectrum remain dependent upon others for care and support for their entire lives, with limited relationships and social functioning.

Results from studies over time, into the later development and transition in adulthood confirm that there remained a chronicity of social impairment and challenging behaviour in those with autism and learning disabilities (Wing and Gould 1979).

BIOLOGICAL CAUSES OF AUTISM

There are many current theories with no certainty, and several that have been refuted, such as the now rejected environmental theory - Bettelheim's (1967) "Frigid Mother Syndrome". The current theories fall into two broad categories - psychological (1) and biological, although the strongest current evidence supports biological theories (MRC 2001).

Evidence for an Organic Cause

Studies have found that as much as 90% of samples had evidence of brain damage or dysfunction (Grecius 2003). Olsson et al (1988) suggested that one indication that brain damage is at the root of autism is the high incidence of epilepsy in autistic children.

While Smalley et al (1988) noted that people with progressively lower IQ increases the incidence of autism.

Campbell et al (1982) showed with CAT scans that autistic people had brain abnormalities generally.

More specifically, Rumsey and Hamburger (1988) and Ozonoff et al (1991) discussed that the frontal lobes may be implicated in autism. Individuals with autism tend to do poorly on tasks which adults with acquired lesions to frontal lobes also fail.

The areas of brain differences include the cerebellum, limbic system, cerebral cortex, or brain size as a whole, according to recent neuroimaging studies (Grecius 2003).

Evidence for a Hereditary Cause

Folstein and Rutter (1977) found identical (monozygotic) twins had a far higher concordance rate for autism (36%) than dizygotic twins (0%) in their classic study of 21 twin pairs. A genetic component is weighty (up to 90% for MZ twins), although the exact role of the child's genes is far from clear (Rutter 1991). Table 7 lists the results of three key twin studies in autism.

STUDY	CONCORDANCE RATES	
	MZ	DZ
Le Couteur et al (1989)	57%	0%
Steffenberg et al (1989) Nordic	91%	0%
Bailey et al (1995) Britain	69%	0%

Table 7 - Results of three key studies twin studies on autism.

Smalley et al (1988) noted that autism exhibits itself fifty times more frequently in siblings of autistic children.

Modern genetic research techniques suggest the possible role of genes on chromosomes 1, 2, 6, 7, 13, 16, 18, and 19 (MRC 2001) (2). But it is more likely that many genes interact to create the vulnerability to autistic spectrum disorders (MRC 2001).

Fragile X syndrome, and tuberous sclerosis increase the risk of autism (eg: Gillberg and Forsell 1984)

There is no shortage of different biological explanations for autism. Table 8 lists a number of the other ideas being researched in varying degrees currently.

1. Neuroanatomy
- brainstem abnormalities (Rodier 2000)
- lack of hemispheric lateralisation (Mesibov et al 1997)
- enlarged brain volume (Grecius 2003)

2. Biochemistry
- serotonin elevation (Bailey et al 1996)
- opioid excess theory (Kidd 2002b)

3. Metabolic abnormalities (Kidd 2002a)

4. Immune system problems (Phillips 2002)

Table 8 - Some of the different explanations for autism being researched currently.

TREATMENTS

There is currently no cure for the core triad of impairments or suggested biological and cognitive conditions. But medication is prescribed to treat the medical problems associated with it (eg: seizures), and the symptoms such as anxiety, and behavioural problems (eg: hyperactivity, aggression and ritualised behaviour).

However, autistic symptoms, the behaviours, can be treated with varying degrees of success and many are available, based upon the prevailing understanding of the cause of autism. No one treatment has been known to be effective and individuals are often given a combination. They fall under the following two broad headings, psychological and medical.

1. Psychological Treatments

Psychological treatments attempt to eliminate negative behaviours and teach normal behaviours and

coping mechanisms and fall into two categories: behavioural, and psycho-educational (eg: T.E.A.C.C.H - Treatment and Education of Autistic and related Communication Handicapped Children).

a) Behavioural Treatment Methods

These focus upon eliminating "negative" target behaviours that are associated with autism - challenging behaviour like self injury, stereotypical behaviours, and aggression, and promoting more normal patterns of behaviour such as social skills, language and daily living skills.

These types of treatment include:

- positive procedures (praise for "normal behaviour" and ignoring of negative behaviours;
- aversive procedures (punishments upon presentation of negative target behaviour);
- combinations of the above and others.

Their advantages include that they have been empirically evaluated for effectiveness. The best example of this is the Lovaas technique, an early intensive behavioural therapy often up to 40 hours per week. It teaches social/academic and compliance skills using reinforcement methods, from an early age.

Lovass practitioners visit and train parents to interact with the child. The child learns that when X happens they do Y. If they do Y, this is reinforced and becomes part of learned behaviour.

b) Psychoeducational Treatments

Many treatments have developed over the last few years, with little evaluation, including music therapy, dolphin therapy, and facilitated communication. However, some are relatively well evaluated and have produced some measure of success.

Environmental management techniques are some of the most effective and well evaluated; eg: T.E.A.C.C.H. This teaching method is based upon the understanding of the core deficits - the triad - and uses "structured teaching" in a low arousal environment, with clear communication (eg: schedules and timings for events), so the environment is predictable (in words/pictures /objects of reference) (Mesibov et al 1997).

"Teaching Theory of Mind" (Baron-Cohen 1995) focuses on the triad of impairment as resulting from a cognitive deficit of the fundamental human ability to "mind read",

or understand and predict another person's behaviour. This method teaches autistic children to "mind read" using computers and conventional methods.

2. Medications

These are many and varied. Often medication developed for the treatment of psychoses (neuroleptics), to treat anxiety, and stimulants (to increase attention span and reduce excitability and distractability) are prescribed.

Medication is used to treat associated medical problems; eg: epilepsy (anti-convulsants). Neuroleptics are also prescribed; eg: chlorpromazine or haliperidol. Chlorpromazine has a high sedative effect. Haliperidol does produce good results in both the short and long term, improving co-ordination and self-care, and reducing stereotypical behaviours, withdrawal, hyperactivity and figitiness. However, adverse side effects including weight gain are extreme.

Antidepressants (eg: SSRIs like fluoxetine) are effective in reducing self injurious behaviour, but again the side effects are extreme - dry mouth, nausea, constipation and urinary retention (Coghill 2002).

Stimulants, again often used (eg methylphenidate and dextramphetamine), lead to emergence of other behaviours if not already there, such as increased stereotypical behaviours. There are reports of good outcomes for people with high functioning autism (though still being evaluated).

CONCLUSIONS

Current research into both causes and treatments is widespread and does appear to offer some hope for those with autism and their families and carers, as do ongoing programmes.

The Autism Research Unit in Sunderland is looking into chemical causes of autism and the use of gluten free diets to alleviate the symptoms of autism (eg: Kidd 2002b). The Autism Research Unit in Cambridge investigates the cognitive, genetic and neural features of autism; eg: Bailey and colleagues (1996) research into genetic causes.

Inconclusive studies into MMR vaccine have prompted petitions to the Department of Health to study the safety of the triple vaccine (discussed in Tidmarsh 2003).

Reviews of current research and ideas are found in MRC (2001), Lord and Bailey (2002), and Grecius (2003).

FOOTNOTES

1. Psychological theories tend to have focused upon the cognitive deficits associated with autism (Bailey et al 1996; Baron-Cohen 1997).

2. Details of methods of modern genetic research in "Biological Psychiatry", Psychology Teachers Update no.3, May 2003.

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